Cuba
AUTHOR EMAIL: delegcha@ceniai.cu
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CODEN: HPSRA ISSN: 0138-7103
DOCUMENT TYPE: Journal; Article
LANGUAGE: SPANISH SUMMARY LANGUAGE: ENGLISH; SPANISH
NUMBER OF REFERENCES: 61

...the understanding of the physiological role of nerve growth factor (NGF) have raised the question of whether the neurotrophins might have clinical potential in the *treatment* of *neurodegenerative* *disease*, nerve trauma nd value their possible relationship in the psychosis. Although NGF was first characterized as a target-derived survival factor for developing sympathetic and...

...for other neurotrophins that might act on the many classes of neurons that do not respond to NGF. The neurotrophin family includes NGF, brain derived *neurotrophic* *factor* (*BDNF*), neurotrophin-3 (NT- 3) and NT-4/5. A family of related high-affinity receptors for neurotrophins have been identified and are termed trkA, trkB and trkC. TrkA is the receptor for NGF, trkB for *BDNF* and NT-4/5, and trkC is the NT-3 receptor. In addition a 75kDa low affinity (p75) NGF receptor exists which binds each of the neurotrophins and appears to play a role in modulating biological responses mediated by the high-affinity receptors. In this *review*, the biology of the recently discovered family of neurotrophins and their receptors and clinical trial are reviewed, specially in the context of the therapeutic potential (Trophic *Therapy*) of these factors in the *treatment* of neurological disorders.

24/3,K/2 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

07176413 EMBASE No: 1998067908
The preclinical rationale for the use of insulin-like growth factor-I in amyotrophic lateral sclerosis
Festoff B.W.
Dr. B.W. Festoff, Neurobiology Res. Laboratory (151), Dept Veterans Affairs Medical Center, 401 Linwood Blvd, Kansas City, MO 64128 United States
Drugs of Today (DRUGS TODAY) (Spain) 1998, 34/1 (65-77)
CODEN: MDACA ISSN: 0025-7656
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 78

This *review* details the general physiology, biochemistry and molecular biology of insulin-like growth factor I (IGF-I), a pleiotropic factor, and the only one to date showing beneficial effects in a prototypic *neurodegenerative* *disease*, amyotrophic lateral sclerosis (ALS). The preclinical rationale for IGF-I use in treating patients with ALS stems from the fact that this molecule has endocrine...

...cleave individual binding proteins that serves to finely adjust the cellular responses to IGF-I. In order to explain why this trophic factor, unlike ciliary *neurotrophic* *factor* (CNTF) and brain-derived *neurotrophic* *factor* (*BDNF*), was found to have efficacy in large-scale clinical trials in ALS patients, evidence is offered that IGF-I affects all components of the motor...
DRUG DESCRIPTORS:
*somatomedin c--clinical trial--ct; *somatomedin c--drug *therapy*--dt protein tyrosine kinase--endogenous compound--ec; cell surface receptor --endogenous compound--ec; binding protein--endogenous compound--ec; proteinase--endogenous compound--ec; ciliary *neurotrophic* *factor*

--endogenous compound--ec; brain derived *neurotrophic* *factor*
--endogenous compound--ec; thrombin--endogenous compound--ec; serine proteinase--endogenous compound--ec; somatomedin binding protein
--endogenous compound--ec; recombinant somatomedin c--clinical trial--ct; recombinant somatomedin c--drug *therapy*--dt; riluzole--drug *therapy*--dt
MEDICAL DESCRIPTORS:
*amyotrophic lateral sclerosis--drug *therapy*--dt
...central nervous system; peripheral nervous system; motoneuron; nerve fiber; neuromuscular synapse; muscle cell; human; nonhuman; animal experiment; animal model; animal cell; clinical trial; meta analysis; *review*
?ds

Set	Items	Description
S1	8982	(NEUROGENESIS) OR (NEURONAL (W) PRODUCTION)
S2	354	S1 AND ((NEUROTROPHIC (W) FACTOR) OR (BDNF) OR (NEUROTROPHIN??))
S3	7	S2 AND (VECTOR OR (GENE (W) THERAPY))
S4	6	RD (unique items)
S5	7	S2 AND (VECTOR)
S6	40	S2 AND (TREATMENT OR THERAPY)
S7	0	S6 AND (NEURODEGENERATIVE (W) CONDITION)
S8	0	S6 AND (HUNTINGTON'S (W) DISEASE)
S9	2	S6 AND (HUNTINGTON)
S10	2	RD (unique items)
S11	27	RD S6 (unique items)
S12	4	S11 AND REVIEW
S13	28	S2 AND REVIEW
S14	24	RD (unique items)
S15	1	S14 AND (HUNTINGTON)
S16	167	(NEURODEGENERATIVE (W) DISEASE) AND (NEUROTROPHIC (W) FACTOR)
S17	11	S16 AND (HUNTINGTON)
S18	3	S17 AND (BDNF)
S19	0	S16 AND ((LATERAL (W) VENTRICLES) OR (VENTRICULAR (W) ZONE-))
S20	105	S16 AND (TREATMENT OR THERAPY)
S21	18	S20 AND REVIEW
S22	14	RD (unique items)
S23	2	S22 AND (BDNF)
S24	2	RD (unique items)

?t s22/3,k/all

22/3,K/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10876028 20435204 PMID: 10978846

Glial cell line-derived *neurotrophic* *factor* (GDNF) as a defensive molecule for *neurodegenerative* *disease* : a tribute to the studies of antonia vernadakis on neuronal-glial interactions.

Bohn M C; Kozlowski D A; Connor B
Children's Memorial Institute for Education and Research, Department of Pediatrics, Children's Memorial Hospital, Northwestern University Medical School, Chicago, IL 60613, USA. m-bohn@nwu.edu

International journal of developmental neuroscience : the official journal of the International Society for Developmental Neuroscience (ENGLAND) Nov 2000, 18 (7) p679-84, ISSN 0736-5748 Journal Code: 8401784
Document type: Journal Article; Review; Review, Tutorial
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

Glial cell line-derived *neurotrophic* *factor* (GDNF) as a defensive molecule for *neurodegenerative* *disease* : a tribute to the studies of antonia vernadakis on neuronal-glial interactions.

Research stemming from interests in neuronal-glial interactions has led to the identification of a number of novel trophic factors, such as the dopaminergic *neurotrophic* *factor* glial cell line-derived *neurotrophic* *factor* (GDNF). Delivery of the GDNF gene to rat models of Parkinson's disease suggests a potential clinical use of GDNF gene *therapy* for humans with this disease. This *review* article briefly summarizes the history of GDNF and the effects of GDNF gene delivery prior to or after a lesion of the rat nigrostriatal system.

Chemical Name: Nerve Growth Factors; Nerve Tissue Proteins; glial cell-line derived *neurotrophic* *factor*

22/3,K/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07969833 94109541 PMID: 8282068

The therapeutic potential of neurotrophic factors in the *treatment* of Parkinson's disease.

Lindsay R M; Altar C A; Cedarbaum J M; Hyman C; Wiegand S J

Regeneron Pharmaceuticals Inc., Tarrytown, New York 10591-6707.

Experimental neurology (UNITED STATES) Nov 1993, 124 (1) p103-18, ISSN 0014-4886 Journal Code: 0370712

Document type: Journal Article; Review; Review, Academic

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

The therapeutic potential of neurotrophic factors in the *treatment* of Parkinson's disease.

...of neurotrophic growth factors, especially members of the nerve growth factor-related neurotrophin family, which may point to their potential as therapeutic agents for the *treatment* of Parkinson's disease. Parkinson's disease, characterized by the progressive loss of dopaminergic neurons of the substantia nigra, is one of the most well...

... yield obvious therapeutic strategies, but even in the absence of such knowledge there are several general approaches that can be taken as strategies for the *treatment* of a "focal" *neurodegenerative* *disease*. These include: (a) mimetics, activation of the postsynaptic target(s) of the missing neurons through mimetics of the missing neurotransmitter, e.g., use of a...

... c) neurotrophic factors or neuroprotectants, intervention with neurotrophic factors/neuroprotective agents which slow, halt, or reverse the progression of neuronal degeneration, e.g., a dopamine *neurotrophic* *factor* in Parkinson's disease. The scope of the present article is limited to a *review* of recent progress in the biology of neurotrophic factors that relates to their potential clinical use in treating the loss of dopamine neurons in Parkinson...

Descriptors: Brain--drug effects--DE; *Growth Substances--therapeutic use --TU; *Nerve Growth Factors--therapeutic use--TU; *Parkinson Disease--drug *therapy*--DT

22/3,K/3 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE

Adams

11370175 EMBASE No: 2001385149

Sustained delivery of GDNF: Towards a *treatment* for Parkinson's disease

Zurn A.D.; Widmer H.R.; Aebischer P.

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Brain Research Reviews (BRAIN RES. REV.) (Netherlands) 2001, 36/2-3 (222-229)

CODEN: BRERD ISSN: 0165-0173
PUBLISHER ITEM IDENTIFIER: S0165017301000984
DOCUMENT TYPE: Journal ; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 60

Sustained delivery of GDNF: Towards a *treatment* for Parkinson's disease

Parkinson's disease (PD) is a *neurodegenerative* *disease* characterized by the progressive loss of nigral dopaminergic neurons. Although symptomatic therapies to substitute for the missing neurotransmitter dopamine are efficient at the early stages of the disease, the goal is to find alternative therapies which could protect dopaminergic neurons from the degenerative process. We have used two distinct gene *therapy* approaches to deliver the neuroprotective molecule glial cell line-derived *neurotrophic* *factor* (GDNF) in animal models of the disease: (i) an encapsulated genetically engineered cell line releasing GDNF (ex vivo gene *therapy*); and (ii) a lentiviral vector encoding the GDNF gene (in vivo gene *therapy*). Both approaches allowed protection of nigral dopaminergic neurons against lesion-induced cell death in rodent as well as monkey models of PD. Behavioral symptoms were...
DRUG DESCRIPTORS:
*glial cell line derived *neurotrophic* *factor*--adverse drug reaction--ae ; *glial cell line derived *neurotrophic* *factor*--clinical trial--ct; * glial cell line derived *neurotrophic* *factor*--drug administration--ad; * glial cell line derived *neurotrophic* *factor*--drug dose--do; *glial cell line derived *neurotrophic* *factor*--drug *therapy*--dt; *glial cell line derived *neurotrophic* *factor*--pharmaceutics--pr; *glial cell line derived *neurotrophic* *factor*--pharmacology--pd; *glial cell line derived *neurotrophic* *factor*--intracerebral drug administration--ce; *glial cell line derived *neurotrophic* *factor*--intracerebroventricular drug administration--cv; *recombinant protein--adverse drug reaction--ae; * recombinant protein--clinical trial--ct; *recombinant protein--drug administration--ad; *recombinant protein--drug dose--do; *recombinant protein--drug *therapy*--dt; *recombinant protein--pharmaceutics--pr; * recombinant protein--pharmacology--pd; *recombinant protein--intracerebral drug administration--ce; *recombinant protein--intracerebroventricular drug administration--cv
MEDICAL DESCRIPTORS:
*Parkinson disease--drug *therapy*--dt; *gene targeting; *neuroprotection cell loss; dopaminergic nerve cell; tissue graft; disease course; hormone substitution; gene *therapy*; Lentivirinae; virus vector; cell line; genetic engineering; disease model; genetic code; cell death; rodent; monkey; animal behavior; nerve cell differentiation; cell survival; protein secretion; drug induced disease--side effect--si; human; nonhuman; clinical trial; *review*; priority journal

22/3,K/4 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

10874440 EMBASE No: 2000360003
Neuroimmunophilin ligands: Evaluation of their therapeutic potential for the *treatment* of neurological disorders
Gold B.G.
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AUTHOR EMAIL: gold@ohsu.edu
Expert Opinion on Investigational Drugs (EXPERT OPIN. INVEST. DRUGS) (United Kingdom) 2000, 9/10 (2331-2342)
CODEN: EOIDE ISSN: 1354-3784
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 102

Neuroimmunophilin ligands: Evaluation of their therapeutic potential for the *treatment* of neurological disorders

Neuroimmunophilin ligands are a class of compounds that hold great promise for the *treatment* of nerve injuries and neurological disease. In contrast to neurotrophins (e.g., nerve growth factor), these compounds readily cross the blood-brain barrier, being orally...

...the limiting side effects produced by these drugs arise via calcineurin inhibition. A major breakthrough for the development of this class of compounds for the *treatment* of human neurological disorders was the ability to separate the neuroregenerative property of FK-506 from its immunosuppressant action via the development of non-immunosuppressant...

...a component of steroid receptor complexes). Thus, steroid receptor chaperone proteins represent novel targets for future drug development of novel classes of compounds for the *treatment* of a variety of human neurological disorders, including traumatic injury (e.g., peripheral nerve and spinal cord), chemical exposure (e.g., vinca alkaloids, Taxol(TM) and *neurodegenerative* *disease* (e.g., diabetic neuropathy and Parkinson's disease).

DRUG DESCRIPTORS:

*immunophilin--clinical trial--ct; *immunophilin--drug analysis--an; *immunophilin--drug development--dv; *immunophilin--drug *therapy*--dt; *immunophilin--pharmacology--pd; *ligand--clinical trial--ct; *ligand--drug analysis--an; *ligand--drug development--dv; *ligand--drug *therapy*--dt; *ligand--pharmacology--pd

calcineurin--endogenous compound--ec; chaperone--endogenous compound--ec; tsukubaenolide--drug analysis--an; tsukubaenolide--drug *therapy*--dt; tsukubaenolide--pharmacology--pd; cyclosporin A--drug *therapy*--dt; cyclosporin A--pharmacology--pd; fk 506 binding protein--drug analysis--an; fk 506 binding protein--drug *therapy*--dt; fk 506 binding protein --pharmacology--pd; geldanamycin--drug analysis--an; geldanamycin --pharmacology--pd; radicicol--drug analysis--an; radicicol--pharmacology --pd; Vinca alkaloid--adverse drug...

...acrylamide--drug toxicity--to; carbon tetrachloride--drug toxicity--to; heat shock protein 90--endogenous compound--ec; fk 506 binding protein --drug development--dv; brain derived *neurotrophic* *factor*--pharmacology --pd; neurotrophin--pharmacology--pd; unclassified drug

MEDICAL DESCRIPTORS:

*neurologic disease--drug *therapy*--dt

neuroprotection; drug mechanism; enzyme inhibition; side effect--side effect--si; nerve regeneration; drug structure; neurotropism; carpal tunnel syndrome--drug *therapy*--dt; nerve transection--drug *therapy*--dt; transplantation; hand surgery; limb; diabetic neuropathy--drug *therapy* --dt; peripheral neuropathy--drug *therapy*--dt; drug induced disease--drug *therapy*--dt; sensory neuropathy--drug *therapy*--dt; dementia--drug *therapy*--dt; spinal cord injury--drug *therapy*--dt; brain injury--drug *therapy*--dt; brain ischemia--drug *therapy*--dt; Alzheimer disease--drug *therapy*--dt; Huntington chorea--drug *therapy*--dt; Parkinson disease --drug *therapy*--dt; autoimmune disease--drug *therapy*--dt; immunosuppressive *treatment*; human; nonhuman; mouse; rat; clinical trial; phase 2 clinical trial; animal experiment; animal model; controlled study; human cell; *review*

DRUG TERMS (UNCONTROLLED): neuroimmunophilin ligand--clinical trial--ct; neuroimmunophilin ligand--drug analysis--an; neuroimmunophilin ligand--drug development--dv; neuroimmunophilin ligand--drug *therapy*--dt; neuroimmunophilin ligand--pharmacology--pd; fkbp 12--drug analysis--an; fkbp 12--drug *therapy*--dt; fkbp 12--pharmacology--pd; fkbp 52--drug analysis--an; fkbp 52--drug *therapy*--dt; fkbp 52--pharmacology--pd; 1 (3,3 dimethyl 1,2 dioxopentyl) 2 pyrrolidinecarboxylic acid 3 (3 pyridyl)propyl ester--drug analysis--an; 1 (3,3 dimethyl 1,2 dioxopentyl) 2 pyrrolidinecarboxylic acid 3 (3 pyridyl)propyl ester--drug *therapy*--dt; 1 (3,3 dimethyl 1,2 dioxopentyl) 2 pyrrolidinecarboxylic acid 3 (3

pyridyl)propyl ester--pharmacology--pd; 1 (3,3 dimethyl 1,2 dioxopentyl) 2 pyrrolidinecarboxylic acid 3 (3 pyridyl)propyl ester--oral drug administration--po; timcodar--clinical trial--ct; timcodar--drug development--dv; timcodar--drug *therapy*--dt; timcodar--pharmacology--pd; nil a--clinical trial--ct; nil a--drug *therapy*--dt; v 13670--drug analysis--an; v 13670--drug development--dv; v 13670--drug *therapy*--dt; v 13670--pharmacology--pd; nt 4--pharmacology--pd

22/3,K/5 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

10639738 EMBASE No: 2000104774
Motor neuron disease - A *review*
Pritchard J.; Swingler R.J.
J. Pritchard, Department of Neurology, Ninewells Hospital, Medical School, Dundee United Kingdom
Scottish Medical Journal (SCOTT. MED. J.) (United Kingdom) 2000, 45/1 (4-7)
CODEN: SMDJA ISSN: 0036-9330
DOCUMENT TYPE: Journal; Note
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 39

Motor neuron disease - A *review*

...to evidence-based guidelines. Whilst scientific advances into the aetiology of MND have been of great importance in both the understanding of this and other *neurodegenerative* *disease*, they have only recently led to *therapy* directed at disease progression. The management of patients with MND remains largely supportive but it is hoped that the future may hold better prospects for...
DRUG DESCRIPTORS:
acetylcysteine--drug *therapy*--dt; alpha tocopherol--drug *therapy*--dt; brain derived *neurotrophic* *factor*--drug *therapy*--dt; ciliary *neurotrophic* *factor*--drug *therapy*--dt; diazepam--drug *therapy*--dt; fentanyl--drug *therapy*--dt; riluzole--drug *therapy*--dt; selegiline --drug *therapy*--dt; somatomedin C--drug *therapy*--dt; xaliproden--drug *therapy*--dt; xaliproden--oral drug administration--po
MEDICAL DESCRIPTORS:
*motor neuron disease--congenital disorder--cn; *motor neuron disease --diagnosis--di; *motor neuron disease--drug *therapy*--dt; *motor neuron disease--epidemiology--ep; *motor neuron disease--etiology--et; *motor neuron disease--*therapy*--th
clinical feature; degenerative disease--congenital disorder--cn; degenerative disease--diagnosis--di; degenerative disease--drug *therapy* --dt; degenerative disease--epidemiology--ep; degenerative disease --etiology--et; degenerative disease--*therapy*--th; disease course; genetic disorder--congenital disorder--cn; genetic disorder--diagnosis--di; genetic disorder--drug *therapy*--dt; genetic disorder--epidemiology--ep; genetic disorder--etiology--et; genetic disorder--*therapy*--th; nervous system; palliative *therapy*; pathogenesis; survival; human; clinical trial ; meta analysis; note

22/3,K/6 (Item 4 from file: 73)
DIALOG(R)File 73:EMBASE

07895199 EMBASE No: 1999368689
Experimental models of amyotrophic lateral sclerosis
Elliott J.L.
J.L. Elliott, Department of Neurology, University of Texas, Southwestern Medical Center, 5323 Harry Hines Boulevard, Dallas, TX 75235 United States

Neurobiology of Disease (NEUROBIOL. DIS.) (United States) 1999, 6/5 (310-320)
CODEN: NUDIE ISSN: 0969-9961
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 99

Amyotrophic lateral sclerosis (ALS) is a chronic *neurodegenerative* *disease* characterized by the progressive loss of motor neurons, leading to profound weakness and eventual death of affected individuals. For the vast majority of patients with ALS, the etiology of the disorder is unknown, and although multiple clinical trials of various therapeutic agents have been undertaken, truly effective *therapy* is not currently available for the disease. The selection of treatments used in ALS clinical trials frequently has its basis in promising data obtained from... ...the proposed agent has shown some effect in protecting motor neurons from a particular insult. The likelihood of a successful clinical outcome for a given *treatment* in ALS would therefore depend on two principal factors, including the similarity of the model to the disease and the biologic action of the potential therapeutic agent. Partly because early experimental models of ALS failed to replicate the disease process, *treatment* success in these models did not carry over into human trials. Recently, however, a variety of newer model systems have been developed and utilized to...

...spinal cord organotypic slice subjected to glutamate excitotoxicity as a model system to test the effectiveness of neurotrophic factors in preventing motor neuron degeneration. This *review* will assess the strengths and weaknesses of differing ALS model systems that have been used to preclinically test potential drug efficacy in ALS.
DRUG DESCRIPTORS:
glutamic acid; *neurotrophic* *factor*--drug *therapy*--dt
MEDICAL DESCRIPTORS:
*amyotrophic lateral sclerosis--drug *therapy*--dt; *amyotrophic lateral sclerosis--etiology--et
experimental model; neuroprotection; drug activity; *treatment* outcome; disease course; nerve cell degeneration; motoneuron; drug efficacy; nonhuman; mouse; rat; animal experiment; animal model; animal tissue; *review*; priority journal

22/3,K/7 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

07487954 EMBASE No: 1998277317
Heterogeneity of the psychoses: Is there a neurodegenerative psychosis?
Knoll IV J.L.; Garver D.L.; Ramberg J.E.; Kingsbury S.J.; Croissant D.; McDermott B.
Dr. D.L. Garver, Dallas VA Medical Center (116A), 4500 S. Lancaster Rd., Dallas, TX 75216 United States
Schizophrenia Bulletin (SCHIZOPHR. BULL.) (United States) 1998, 24/3 (365-379)
CODEN: SCZBB ISSN: 0586-7614
DOCUMENT TYPE: Journal; Article
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 98

...domains of schizophrenia, we demonstrate that currently available data on schizophrenia patients are consistent with the hypothesis that some of these patients have an ongoing *neurodegenerative* *disease*, whereas others do not. We *review* studies (longitudinal and cross-sectional) documenting progressive increases in ventricular size, accelerated loss of brain tissues, progressive delays in *treatment* response, and neurochemical (magnetic resonance spectroscopy) and neurophysiological (P300) indices, all of which are consistent with ongoing cerebral

degeneration in a significant subgroup of schizophrenia...
DRUG DESCRIPTORS:
glutamic acid--endogenous compound--ec; *neurotrophic* *factor*--endogenous compound--ec

22/3,K/8 (Item 6 from file: 73)
DIALOG(R)File 73:EMBASE

07298854 EMBASE No: 1998197299
The relationship among the neurotrophins, Parkinson's disease, Alzheimer's disease and schizophrenia
LA RELACION ENTRE LAS NEUROTROFINAS Y LAS ENFERMEDADES DE PARKINSON, ALZHEIMER Y ESQUIZOFRENIA
Rodriguez J.C.G.; Oviedo H.H.; Gonzalez J.C.P.
J.C.G. Rodriguez, Avenida 17.5008 e/ 50 y 52, Playa, Ciudad de La Habana Cuba
AUTHOR EMAIL: delegcha@ceniai.cu
Revista del Hospital Psiquiatrico de la Habana (REV. HOSP. PSIQUIATR. HABANA) (Cuba) 1997, 18/2 (152-166)
CODEN: HPSRA ISSN: 0138-7103
DOCUMENT TYPE: Journal; Article
LANGUAGE: SPANISH SUMMARY LANGUAGE: ENGLISH; SPANISH
NUMBER OF REFERENCES: 61

...the understanding of the physiological role of nerve growth factor (NGF) have raised the question of whether the neurotrophins might have clinical potential in the *treatment* of *neurodegenerative* *disease*, nerve trauma nd value their possible relationship in the psychosis. Although NGF was first characterized as a target-derived survival factor for developing sympathetic and...

...for other neurotrophins that might act on the many classes of neurons that do not respond to NGF. The neurotrophin family includes NGF, brain derived *neurotrophic* *factor* (BDNF), neurotrophin-3 (NT- 3) and NT-4/5. A family of related high-affinity receptors for neurotrophins have been identified and are termed trkA...

...exists which binds each of the neurotrophins and appears to play a role in modulating biological responses mediated by the high-affinity receptors. In this *review*, the biology of the recently discovered family of neurotrophins and their receptors and clinical trial are reviewed, specially in the context of the therapeutic potential (Trophic *Therapy*) of these factors in the *treatment* of neurological disorders.

22/3,K/9 (Item 7 from file: 73)
DIALOG(R)File 73:EMBASE

07180435 EMBASE No: 1998071668
Fetal transplantation for the *treatment* of neurodegenerative diseases. Current status and future potential
Kopyov O.V.; Jacques S.; Eagle K.S.
Dr. O.V. Kopyov, The Neurosciences Institute, Good Samaritan Hospital, 637 South Lucas Avenue, Los Angeles, CA 90017 United States
AUTHOR EMAIL: okopyov@goodsam.org
CNS Drugs (CNS DRUGS) (New Zealand) 1998, 9/2 (77-83)
CODEN: CNDRE ISSN: 1172-7047
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 78

Fetal transplantation for the *treatment* of neurodegenerative diseases. Current status and future potential

Patients with neurodegenerative diseases currently have few *treatment* options. However, neurotransplantation represents one potential *treatment* avenue. Animal studies using lesion-induced models of neurodegenerative disorders such as Parkinson's disease, Huntington's disease and Alzheimer's disease have shown that...
...symptoms of neurodegenerative diseases include gene transfer or administration of neurotrophic factors, either through direct infusion or by transplantation of biological sources. Currently, however, these *treatment* strategies are still under development and have not been assessed clinically. Continued refinement of the technique of neurotransplantation, possibly in combination with these alternative approaches, promises steady improvement in the *treatment* options for patients with *neurodegenerative* *disease*.
DRUG DESCRIPTORS:
neurotrophic *factor*--drug *therapy*--dt
MEDICAL DESCRIPTORS:
*fetal tissue transplantation; *nerve degeneration--drug *therapy*--dt; * nerve degeneration--surgery--su; *nerve degeneration--*therapy*--th
...disease--surgery--su; huntington chorea--surgery--su; alzheimer disease; motor activity; cognitive defect--surgery--su; disease course; gene transfer; drug infusion; human; nonhuman; clinical trial; *review*; priority journal

22/3,K/10 (Item 8 from file: 73)
DIALOG(R)File 73:EMBASE

07176413 EMBASE No: 1998067908
The preclinical rationale for the use of insulin-like growth factor-I in amyotrophic lateral sclerosis
Festoff B.W.
Dr. B.W. Festoff, Neurobiology Res. Laboratory (151), Dept Veterans Affairs Medical Center, 401 Linwood Blvd, Kansas City, MO 64128 United States
Drugs of Today (DRUGS TODAY) (Spain) 1998, 34/1 (65-77)
CODEN: MDACA ISSN: 0025-7656
DOCUMENT TYPE: Journal; Review
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH
NUMBER OF REFERENCES: 78

This *review* details the general physiology, biochemistry and molecular biology of insulin-like growth factor I (IGF-I), a pleiotropic factor, and the only one to date showing beneficial effects in a prototypic *neurodegenerative* *disease*, amyotrophic lateral sclerosis (ALS). The preclinical rationale for IGF-I use in treating patients with ALS stems from the fact that this molecule has endocrine...

...cleave individual binding proteins that serves to finely adjust the cellular responses to IGF-I. In order to explain why this trophic factor, unlike ciliary *neurotrophic* *factor* (CNTF) and brain-derived *neurotrophic* *factor* (BDNF), was found to have efficacy in large-scale clinical trials in ALS patients, evidence is offered that IGF-I affects all components of the...
DRUG DESCRIPTORS:
*somatomedin c--clinical trial--ct; *somatomedin c--drug *therapy*--dt protein tyrosine kinase--endogenous compound--ec; cell surface receptor --endogenous compound--ec; binding protein--endogenous compound--ec; proteinase--endogenous compound--ec; ciliary *neurotrophic* *factor* --endogenous compound--ec; brain derived *neurotrophic* *factor* --endogenous compound--ec; thrombin--endogenous compound--ec; serine proteinase--endogenous compound--ec; somatomedin binding protein --endogenous compound--ec; recombinant somatomedin c--clinical trial--ct; recombinant somatomedin c--drug *therapy*--dt; riluzole--drug *therapy*--dt
MEDICAL DESCRIPTORS:

*amyotrophic lateral sclerosis--drug *therapy*--dt
...central nervous system; peripheral nervous system; motoneuron; nerve fiber; neuromuscular synapse; muscle cell; human; nonhuman; animal experiment; animal model; animal cell; clinical trial; meta analysis; *review*

22/3,K/11 (Item 9 from file: 73)
DIALOG(R)File 73:EMBASE

06917362 EMBASE No: 1997201807
Therapeutic advances in amyotrophic lateral sclerosis
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Amyotrophic lateral sclerosis (ALS) is a progressive and rapidly fatal *neurodegenerative* *disease* in which both upper and lower motoneurones are involved. The recent discovery of mutations affecting the superoxide dismutase (SOD) gene has given impetus to research...

...of oxidative stress in the pathogenesis of familial ALS, while further evidence for a role of excitotoxicity in the disease process has arisen. In this *review*, Erik Louvel, Jacques Hugon and Adam Doble discuss these findings and, in addition, describe how a number of large, well-controlled clinical trials have taken...

...by different aetiological hypotheses, including immunosuppressive therapies, neurotrophic factors, antioxidants and anti-excitotoxic drugs. These trials have led to the first modest steps in the *treatment* of this devastating neurological disease.
DRUG DESCRIPTORS:
*brain derived *neurotrophic* *factor*--clinical trial--ct; *brain derived *neurotrophic* *factor*--pharmacology--pd; *brain derived *neurotrophic* *factor*--drug *therapy*--dt; **neurotrophic* *factor*--pharmacology--pd; **neurotrophic* *factor*--clinical trial--ct; **neurotrophic* *factor*--drug *therapy*--dt; *recombinant ciliary *neurotrophic* *factor*--clinical trial --ct; *recombinant ciliary *neurotrophic* *factor*--drug *therapy*--dt; * recombinant ciliary *neurotrophic* *factor*--pharmacology--pd; *recombinant growth hormone--clinical trial--ct; *recombinant growth hormone --pharmacology--pd; *recombinant growth hormone--drug *therapy*--dt; * recombinant somatomedin c--pharmacology--pd; *recombinant somatomedin c --drug *therapy*--dt; *recombinant somatomedin c--clinical trial--ct acetylcysteine--pharmacology--pd; acetylcysteine--drug *therapy*--dt; acetylcysteine--clinical trial--ct; cyclosporin--clinical trial--ct; cyclosporin--drug *therapy*--dt; cyclosporin--pharmacology--pd; dextromethorphan--pharmacology--pd; dextromethorphan--clinical trial--ct; dextromethorphan--drug *therapy*--dt; gabapentin--drug *therapy*--dt; gabapentin--pharmacology--pd; gabapentin--clinical trial--ct; lamotrigine --pharmacology--pd; lamotrigine--clinical trial--ct; lamotrigine--drug *therapy*--dt; nimodipine--pharmacology--pd; nimodipine--drug *therapy*--dt ; nimodipine--clinical trial--ct; riluzole--clinical trial--ct; riluzole --pharmacology--pd; riluzole--drug *therapy*--dt; threonine--pharmacology --pd; threonine--drug *therapy*--dt; threonine--clinical trial--ct; verapamil--pharmacology--pd; verapamil--clinical trial--ct; verapamil--drug *therapy*--dt; unclassified drug
MEDICAL DESCRIPTORS:
*amyotrophic lateral sclerosis--etiology--et; *amyotrophic lateral sclerosis--radiotherapy--rt; *amyotrophic lateral sclerosis--drug *therapy*

--dt
clinical trial; gene mutation; human; immunosuppressive *treatment*; irradiation; lymphoid tissue; motoneuron; nerve degeneration; oxidative stress; pathogenesis; priority journal; *review*; *treatment planning*
DRUG TERMS (UNCONTROLLED): sr 57746a--drug *therapy*--dt; sr 57746a --clinical trial--ct; sr 57746a--pharmacology--pd

22/3,K/12 (Item 10 from file: 73)
DIALOG(R)File 73:EMBASE

06587080 EMBASE No: 1996251705
Parkinson's disease: Biology and aetiology
Temlett J.A.
Neurology Unit, Department of Medicine, Univ. Witwatersrand Medical School, 7 York Road,Parktown, 2193 Johannesburg South Africa
Current Opinion in Neurology (CURR. OPIN. NEUROL.) (United Kingdom) 1996, 9/4 (303-307)
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...factors or combinations of these detrimental factors. Neurochemical imbalances result both in the substantia nigra and neostriatum, resulting in compensatory mechanisms that make this chronic *neurodegenerative* *disease* difficult to evaluate. Acute parkinsonism models have limitations when compared with chronic disease states, and caution should be present when comparing 'parkinsonism' data with human disease. Better understanding of classical neurotransmitters, neuroastive peptides and neurotrophic factors, will hopefully lead to more rational *treatment* approaches, cellular support strategies, and an understanding of the causes of this disease. Glial derived *neurotrophic* *factor* looks the most promising neurotrophic candidate so far tested in culture and in vivo. The result of clinical trials utilizing neurotrophic factors, both as mesencephalic implant support strategies and as definitive *treatment* of idiopathic Parkinson's disease, are awaited with cautious optimism.
DRUG DESCRIPTORS:
free radical--drug toxicity--to; growth factor--endogenous compound--ec; neurotransmitter--endogenous compound--ec; *neurotrophic* *factor* --clinical trial--ct; *neurotrophic* *factor*--endogenous compound--ec; peptide--endogenous compound--ec
MEDICAL DESCRIPTORS:
apoptosis; cell death; degenerative disease--etiology--et; dopaminergic nerve cell; glia cell; human; mitochondrial respiration; neostriatum; oxidative stress *review* substantia nigra

22/3,K/13 (Item 11 from file: 73)
DIALOG(R)File 73:EMBASE

05721686 EMBASE No: 1994130589
Neurotrophic factors: From molecule to man
Lindsay R.M. Wiegand S.J. Altar C.A. DiStefano P.S.
Regeneron Pharmaceuticals Inc., 777 Old Saw Mill River Road,Tarrytown, NY 10591 United States
Trends in Neurosciences (TRENDS NEUROSCI.) (United Kingdom) 1994, 17/5 (182-190)
CODEN: TNSCD ISSN: 0166-2236
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LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

...the understanding of the physiological role of nerve growth factor (NGF) have raised the uestion of whether neurothrophic factors might have clinical potential in the *treatment* of *neurodegenerative* *disease* or